What is noise-induced hearing loss?

ABSTRACT

Noise-induced hearing loss is sensory deafness caused by long-term exposure of the auditory system to a noisy environment. Auditory fatigue is an early symptom of noise-induced hearing loss, and hearing can gradually recover after people leave a noisy environment. However, if people remain in a noisy environment for a prolonged period of time, their hearing will be permanently impaired. Societal changes mean that people are more likely to be exposed to noise. The hearing loss and tinnitus caused by noise seriously affect people's quality of life and lead to huge economic loss.

The pathogenesis of noise-induced hearing loss is complex. Various theories try to explain this, such as the oxidative stress theory, but none perfectly explains the occurrence of noise-induced hearing loss. There is no treatment which can completely reverse the damage. More research is required to explore the pathogenesis and to better guide clinical practice. Preventative strategies, such as educating the public about hearing health, should be adopted to reduce the harm of noise-induced hearing loss.

Noise-induced hearing loss is sensorineural deafness caused by long-term noise exposure. It is the second most common cause of acquired hearing loss after presbyacusis in many countries. The World Health Organization (2017) estimates that approximately 360 million people worldwide suffer from severe hearing loss and approximately 1.1 billion young people (aged between 12 and 35 years old) face hearing loss as a result of noise (Chadha and Cieza, 2017). This article reviews the features and occurrence of noise-induced hearing loss to guide prevention, treatment and future management.

Epidemiology of noise-induced hearing loss

Societal changes are increasing exposure to noise. Although the sensitivity of each individual is different, sound intensity over 85 dB can cause noise-induced hearing loss. High levels of noise exposure usually come from occupational noise (such as factories) or recreational noise (such as personal music players). More and more young people are at risk of noise-induced hearing loss because of the increasing use of headphones to listen to music. Imam and Hannan (2017) reported that people exposed to sound exceeding 89 dB for more than 5 hours per week can suffer permanent hearing damage over time.

Besides loud noise, there are many other risk factors (modifiable and non-modifiable) which can induce progression of noise-induced hearing loss. Modifiable risk factors include smoking, diabetes and lack of exercise, and non-modifiable risk factors include aging, race and genetics. These factors can overlap with noise and accelerate the occurrence of noise-induced hearing loss (Daniel, 2007). Different genders respond almost equally to noise, but gender influences acoustic risk-taking behaviours: boys engage in significantly more high-risk noise activities than girls (Warner-Czyz and Cain, 2016). Older people and those who have ever suffered from sensorineural hearing loss are more susceptible to noise. Approximately 23% of those between the ages of 65 and 75 years suffer from mild or severe hearing loss. Over the age of 75 years, about 40% have hearing impairment (Daniel, 2007). A decibel chart is shown in *Table 1*.

Table 1. Decibel chart

Decibel level (dB)	Source
0	Quietest sound audible
30	Whisper
50–65	Normal conversation
80–85	City traffic noise
95–110	Motorcycle
110–120	Nightclub
110–140	Rock concerts
150	Firecracker

From Daniel (2007)

Noise-induced hearing loss and acute acoustic trauma

The main symptom of noise-induced hearing loss is progressive hearing loss. Transient and moderate noise

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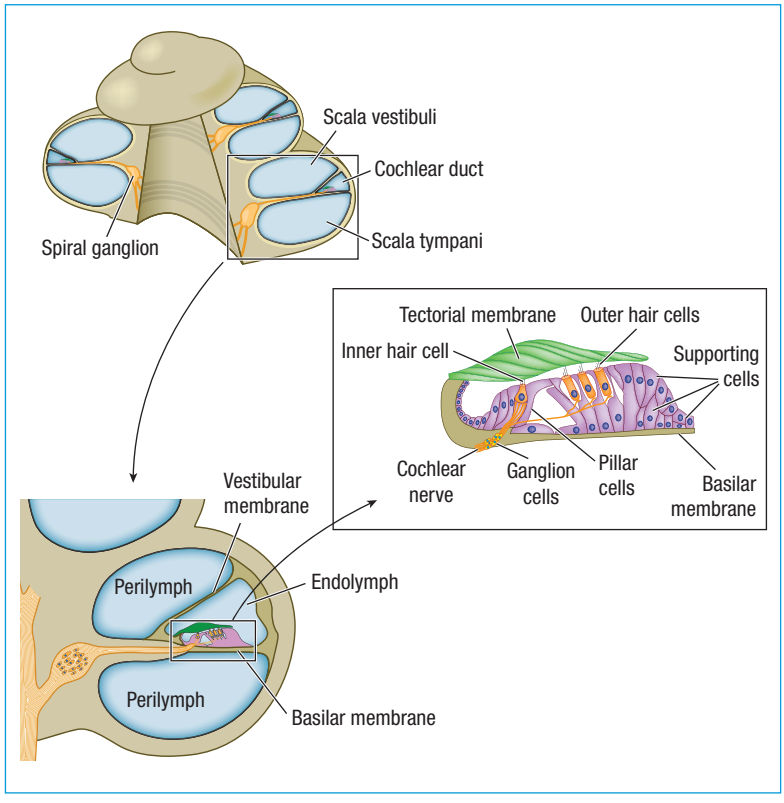

Figure 1. Anatomy of the cochlea.

exposure can lead to temporary threshold shift and hearing can recover after several hours or days (Shi et al, 2015). If medical staff do not intervene in time, or people continue to be exposed to harmful noise, then long-term high-dose noise exposure can cause permanent hearing loss as a result of the death of a large number of the hair cells that perceive sound. High frequency hearing is commonly more susceptible to noise and there is an audiometric notch at around 4 kHz hearing at the early stage of noise-induced hearing loss.

If the noise intensity is extremely high and the duration is very short (such as the sound of a cannon being fired), this can cause acute severe damage of the auditory system called acute acoustic trauma. Noise intensity exceeding 140 dB causes severe deafness and tinnitus immediately after the noise exposure. Acute acoustic trauma is often accompanied by tympanic membrane perforation and ear bleeding after noise damage (Medina-Garin et al, 2016).

Hearing impairment makes it hard for patients to receive sound information, and they may feel silent and depressed. Their speaking skills will also be gradually impaired. Patients are also likely to have chronic and severe symptoms such as tinnitus, which seriously affects daily work and study. Noise can also cause headache, dizziness, insomnia, hypertension and cardiovascular diseases.

Hidden hearing loss and noise-induced hearing loss

Some people have no obvious hearing loss but find it difficult to hear clearly in noisy environments. The hearing acuity and speech discrimination rate of those people is abnormal. This phenomenon is known as 'hidden hearing loss' (Plack et al, 2014). It is difficult for regular hearing tests (such as pure-tone audiometry) to detect hidden hearing loss in time to take steps to prevent further damage. At the early stage of noise-induced hearing loss, noise can cause transient changes in ribbon synapses which are located between hair cells and spiral ganglion neurons. The quantity and quality of ribbon synapses significantly decreases after noise exposure. Although hearing can recover several days later, the number of ribbon synapses does not totally recover (Shi et al, 2015). Various perceptual abnormalities then begin to emerge, including tinnitus and hyperacusis (Kohrman et al, 2019). Long-term noise exposure can lead to continuous apoptosis of hair cells and degeneration of spiral ganglion neurons, which results in the decrease of speech discrimination rate and an increase of the hearing threshold, eventually causing a permanent hearing impairment (Kujawa and Liberman, 2015).

Causes of noise-induced hearing loss

Mechanical damage

It is generally believed that mechanical damage of the cochlea is the main pathological change of noise-induced hearing loss when noise intensity is extremely high. Powerful noise energy is transmitted to the inner ear and causes the perilymph and endolymph to fluctuate violently. The basilar membrane and the tectorial membrane shear and squeeze strongly, which separates the cilia from the inner and outer hair cells, making it difficult for the hair cells to receive effective vibration stimulation. The vibration of the lymph can also separate the hair cells from the basilar membrane, which results in the disruption of ribbon synapses. As a result, the residual synapses cannot maintain optimal function and the encoding ability of hair cells becomes defective. This causes patients to have difficulty in understanding language when they are in a noisy environment (Kujawa and Liberman, 2015; Oxenham, 2016; Van Eynde et al, 2016).

If noise continually acts on the inner ear, then destruction of these inner hair cells (responsible for perceiving the vibration) as well as outer hair cells (responsible for amplifying the sound) can cause sensorineural hearing loss (*Figure 1*). Hair cells are highly differentiated cells which are not able to regenerate (Medina-Garin et al, 2016), so preventing strong noise exposure is very important and should be highly emphasized.

Metabolic damage

Oxidative stress damage

Noise exposure can cause the contraction of cochlear blood vessels and disorders of cell energy metabolism, which generates large amounts of free radicals, such as reactive oxygen species. Dilatation of vessels of the inner ear can also generate free radicals, known as ischaemia–

reperfusion injury. Reactive oxygen species can attack fatty acids in biological membranes (e.g. cell, lysosome or mitochondrial membranes) and produce more free radicals, can attack DNA, causing gene mutation and protein denaturation, and can also increase the expression of apoptotic genes, ultimately leading to cell apoptosis (Kurabi et al, 2017).

Levels of antioxidant enzymes, such as glutathione, can increase in the cochlea after noise exposure. Glutathione is an effective antioxidant and also scavenges free radicals in the cochlea. In addition, after noise exposure, antioxidant enzymes SOD1 gene and HO-1 gene can transcript and translate in large quantities (Fetoni et al, 2015; Honkura et al, 2016). Tuerdi et al (2017) used Mn-SOD knockout mice to observe the effects of partial deletion of endogenous antioxidants, and found that the knockout mice had more severe hearing loss than wild type mice. Although levels of free radicals still increase substantially after noise exposure, the expression of endogenous antioxidants has a certain protective effect, which suggests that the effect of loud noise can be alleviated by supplementing exogenous antioxidants.

Ca^{2+} overload

Noise exposure can cause a large number of voltage-dependent Ca^{2+} channels to open, leading to an influx of Ca^{2+} to hair cells. Noise-induced hearing loss is closely related to excess Ca^{2+} levels because these can trigger reactive oxygen species-independent apoptotic pathways. A high concentration of Ca^{2+} in cells can activate calpain. Activated calpain can decompose cytoskeletal proteins and hydrolyse important proteins such as hormone receptors; it can also activate intracellular protease phospholipase to induce apoptosis. Excessive Ca^{2+} is very harmful to hair cells. Yamaguchi et al (2017) showed that the use of calpain inhibitor can alleviate damage to cytoskeletal proteins and reduce the severity of noise-induced hearing loss by inhibiting the production of calpain and reactive oxygen species. Ca^{2+} channel blockers have protective effects on noise-induced hearing loss. For example, Ye et al (2016) found that perfusing the cochlea with nifedipine-containing perilymph fluid can significantly alleviate noise-induced hearing loss.

Immune and inflammation damage

The inner ear has a strong immune capacity. The cochlea not only contains non-immune cells that can produce immunoregulatory molecules, but also contains many types of immune cells which can be involved in the immune and inflammatory reaction of the inner ear (Fujioka et al, 2014; Wood and Zuo, 2017). After noise exposure, cochlear immune and inflammatory reactions occur within 1–2 days, peak in 3–7 days, and then disappear slowly. The hearing loss is most severe after a few hours, while the immune and inflammatory reactions are greatest after 3–7 days, so hearing loss is not solely caused by the immune and inflammatory reaction (Wood and Zuo, 2017).

> “After noise exposure, cochlear immune and inflammatory reactions occur within 1–2 days, peak in 3–7 days, and then disappear slowly.”

Using high-throughput sequencing RNA-seq technology, Yang et al (2015) found that the expression of immune and inflammatory factors increased throughout the cochlea after noise exposure. Vethanayagam et al (2016) used a toll-like receptor 4 (Tlr4) gene knockout mice model and found that loss of Tlr4 function inhibits the expression of major histocompatibility complex class II, an antigen-presenting molecule. They believed that Tlr4 could regulate multiple aspects of the immune response in the cochlea. Therefore, regulation of the immune and inflammatory reaction may have a positive effect on noise-induced hearing loss. Maeda et al (2018) confirmed that glucocorticoids have a therapeutic effect on noise-induced hearing loss.

Genetic factors

Currently, more than 200 genes related to deafness have been found, but only a few are associated with noise-induced hearing loss. It has been clearly demonstrated that mice carrying certain genes are susceptible to noise. C57BL/6J mice show age-related hearing loss which is caused by genetic defects, and are more likely to develop noise-induced hearing loss than other strains of mice. However, discovering genes related to susceptibility to noise-induced hearing loss in humans is very difficult, because it is almost impossible to collect a group of subjects who have been exposed to the same noise conditions. Consequently, researchers began to screen for single nucleotide polymorphisms. Although most mutations do not affect normal gene function, if point mutations occur in the gene sequence or in the gene regulatory sequence, the function of the gene is bound to be affected.

Genes related to oxidative stress

Free radicals are eliminated by the body's antioxidant defence systems, such as superoxide dismutase. The deletion or mutation of related genes will inevitably affect the auditory function. NRF2, a transcriptional activator, is a key target for prevention of noise-induced hearing loss. One human single nucleotide polymorphism that reduces the transcription NRF2 gene was significantly associated with hearing loss among people subjected to occupational noise exposure (Honkura et al, 2016). Zong et al (2019) showed that mutation or deletion of the glutathione sulfotransferase gene may lead to noise-induced hearing loss in the Chinese Han population and aggravate noise-induced hearing loss when it is combined with smoking.

Genes related to K^+ metabolism

The K^+ metabolism genes are important to the auditory system. Studies have confirmed that multiple mutations of K^+ metabolism genes can lead to the development of

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noise-induced hearing loss, such as KCNE1 (Guo et al, 2018). Furthermore, Swedish scholars found that 35 single nucleotide polymorphisms of K^+-related genes are involved in the regulation of the K^+ channel in the inner ear: among these, three single nucleotide polymorphisms of KCNE1 gene, one single nucleotide polymorphism of the gated voltage K^+ channel gene and one single nucleotide polymorphism of the delayed rectifier K^+ current channel gene are closely related to noise-induced hearing loss.

Heat shock protein genes

Heat shock proteins are a group of conserved proteins that contribute to the synthesis, folding, assembly and transport of many other proteins in the cells. Heat shock proteins are widely expressed in cells and their expression can increase under stress conditions, such as noise exposure. Three genes are involved in heat shock protein synthesis in humans: HSP70-1, HSP70-2 and HSP70-hom. Mutations in these genes are associated with susceptibility to noise-induced hearing loss (Sliwinska-Kowalska and Pawelczyk, 2013; Lei et al, 2017).

Genes that encode specific proteins

The genes encoding procadherin (PCDH15) and myosin (MYH14) are associated with noise-induced hearing loss. Mutation of these genes can profoundly affect the mechanical–electrical transduction pathway, which causes hearing impairment and tinnitus (Sliwinska-Kowalska and Pawelczyk, 2013; Zhang et al, 2014). A mutation in the gene encoding an ATP-gated P2X2 receptor is also related to noise-induced hearing loss. This protein regulates various physiological responses, such as excitatory postsynaptic potential and the mechanical–electrical conduction pathway (Yan et al, 2013).

Other reasons

Noise exposure not only has a direct impact on the auditory system, but also causes physiological and psychological stress. Noise can activate the hypothalamic–pituitary–adrenal axis which regulates the sensitivity of the auditory system. If the cochlea lacks adrenocorticotropin releasing factor receptors (the key factor in hypothalamic–pituitary–adrenal function), the hormone balance in mice is disturbed and these mice are more susceptible to noise exposure (Vetter, 2015). Humans may also experience similar physiological and psychological responses when exposed to noise.

Prevention and treatment

Because noise-induced hearing loss can not be completely cured, preventive measures should be emphasized and taken to alleviate the severity of noise-induced hearing loss. For example, people should stay away from noisy environments and listen to loud music through headphones as little as possible. The source and intensity of noise also needs to be curbed and harnessed by governments. When people work in factories that generate a lot of noise, employers must monitor noise levels and protect workers' hearing, for example by providing anti-sound earplugs. People who have suffered from sensorineural deafness should avoid excessive noise contact. As recreational noise is also an important source of noise pollution, hearing education programmes targeting young people should be encouraged because such education can change people's listening habits and thus protect their hearing (Keppler et al, 2015).

Given the pathogenesis of noise-induced hearing loss, some drugs could alleviate and partially treat noise-induced hearing loss. Potential treatment options include some exogenous antioxidants, calcium antagonists or glucocorticoids, which can reduce the impact of noise after noise exposure through different pathways. Because the pathogenesis of noise-induced hearing loss is very complex and human hair cells do not regenerate, the treatment of noise-induced hearing loss is very difficult and various treatment strategies should be combined. At the early stage, inner ear damage is partially recoverable, so doctors should positively intervene in this phase of noise-induced hearing loss. People who suffer from severe deafness in the late stage of noise-induced hearing loss can only be helped by hearing aids and cochlear implants.

Conclusions

With the continuous development of society, people are more readily exposed to excessive noise and their hearing is being affected. Treatment for noise-induced hearing loss is limited, so people should stay away from noisy environments. Further research and new medicines should be explored. Studies into stem cell differentiation and hair cell regeneration have been promising. If hair cells were able to regenerate, noise-induced hearing loss could hopefully be completely cured (Imam and Hannan, 2017).

Because genetic research into noise-induced hearing loss is still in the exploratory stages, it is inappropriate and difficult to predict the susceptibility of individuals to noise-induced hearing loss by genetic testing at present. People with potential genetic defects are more likely to suffer from tinnitus and decreased speech discrimination rate, and thus should stay away from noise as far as possible. To decrease the incidence of noise-induced hearing loss in the future, increased public awareness is needed. **BJHM**

Conflict of interest: none.

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KEY POINTS

- More and more people worldwide suffer from hearing loss as a result of noise pollution, and young adults and children are at risk of noise-induced hearing loss because of the increasing use of headphones to listen to music.
- Mechanical damage can cause the destruction of ribbon synapses, cilia and hair cells. This affects the perception of sound and results in a decrease in the speech discrimination rate and an increase of the hearing threshold, eventually causing a permanent hearing impairment.
- There are many types of metabolic damage after noise exposure. Free radicals, excess calcium ions and various immune inflammatory factors can affect the normal metabolism of hair cells and cause hearing impairment.
- Although more than 200 genes are related to deafness, only those related to oxidative stress and K^+ metabolism, genes encoding specific proteins and heat shock protein genes are associated with noise-induced hearing loss.
- No treatment can completely reverse the damage caused by noise exposure, so preventative strategies, public awareness about hearing health and early intervention are very important.